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Original Research Article

Impairment of Memory and Novelty of Wistar Albino Rats Exposed to Acute Noise Stress

Sundareswaran Loganathan, Sheeladevi Rathinasamy

Department of Physiology, Dr. ALM. Post Graduate Institute of Basic Medical Sciences, University of Madras, Taramani Campus, Chennai, India.

Corresponding Author: Sheeladevi Rathinasamy

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ABSTRACT

The aim of the study is to elucidate the effect of acute noise stress on memory and novelty. Rats were exposed to acute noise/4 hr/day and analyzed for memory and novelty. The results were noted after noise exposure at the interval of 1 hr, 4 hr and post day and shows a significant alterations in working and reference memory, time to complete in radial maze, novel arm entry, latency period, triad arm errors in Y maze only after 1 hr and not after 4hr and 24hrs. These differences after 1 hr were correlated well with the increase in corticosterone. This study explains the soundless effect of acute noise (unavoidable stressor) impairment on retrieval of memory and novelty which accounts only for a short period of time but it may provide insight to the deleterious effect of repeated noise effect on memory which may be permanent among noise-exposed workers and commonalities.

Key words: Noise stress, working memory, reference memory, novelty, corticosterone, maze

INTRODUCTION

Noise stress is considered as a major threatening problem to human health in this modern world. It is unavoidable and affects the health in all aspects e.g. sleep disturbance (Quis, 1999) hypertension (Chang et al., 2009). Stress is accompanied with the increase in catecholamines and glucocorticoids, which is the main stress hormones (McEwen, 2000). These stress hormones have both protective and damaging effects throughout the body. Acute stress is essential for adaptation and homeostasis maintenance (flight or fight response), whereas chronic stress can cause cognitive and emotional disturbances (McEwen 2007). However, stress is a potent modulator of cognitive function in general, and more precisely, of learning and memory processes (McEwen and Sapolsky 1995). Stress exerts diverse effects on health, emotion, and cognition via catecholamines and glucocorticoids (De Kloet et al.,2005) when goes beyond its normal level, which is essential to regulate and maintain the working memory in humans (Swartz et al., 2008) and rodents (Ramos et al., 2005). Hence, exposure to elevated stress or stress hormone affects learning and memory (McEwen 1999). However, it differs accordingly to the type, duration and intensity of stress. Further, mild stressors are

reported to have distinct effects on cognitive function (Joela et al., 2006). The brain regions are primary targets of stress hormones that include hippocampus (mediating certain types of learning and memory), amygdala (mediating fear responses), and prefrontal cortex (mediating working memory). So the effects of Glucocorticoids and catecholamines that are released during stressful episodes modulate memory processes in the brain areas mainly hippocampus, prefrontal cortex and amygdale (McGaugh, 2008), also findings suggest that simultaneous glucocorticoid and noradrenergic activation changes the patterns of brain activity and may contribute to the differential effects of stress on different memory processes (in particular consolidation and retrieval). So far studies which shown harmful effect of acute stress on learning, memory and cognition were reported by many researchers (Manikandan et al.,2006; Cui et al., 2009; Robinson et al., 2008;Ezlinga et al., 2005;Liston et al., 2006; Yuen et al., 2011; Eunice et al., 2009). On the other hand the encouraging effect on memory in learning performance after acute stress were reported by many scientist (Roozendaal et al., 2006; Creswell et al., 2013;Cordero et al., 2003; Duncko et al., 2009; Vedhara et al., 2000; Kuhlmann, 2005).Studies shows controversial reports of both enhanced and impaired working memory performance (Kuhlmann, S. (2005) improved or not affected (Vedhara, K., 2000). Thus the scope of this study is to resolve the conflicting effects of acute stress on memory, cognition and novelty assessed by eight arm maze and Y maze and acute noise stress exposure were given at the level of retrieval of the memory of Wistar albino rats

MATERIALS AND METHODS

The study was initiated with a proper approval by the Institute's Animal Ethical Committee (IAEC No 01/19/2013). Healthy adult male Wistar rats weighing about 180-200 g have been used for this study. Animals were housed in groups of three (rats) per cage and maintained in a temperature controlled room with a 12-h light: 12-h dark cycle and allowed free access to food and water. All behavioral experiments were likewise carrying out by a human observer in a blind fashion.

Study Groups:

The animals were divided into two groups with six animals in each group

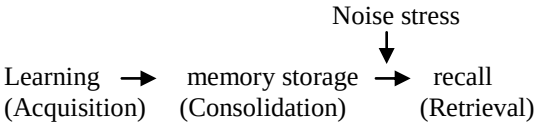
Group I - Control

Group II - Acute noise stress

The control group animals were treated similarly in all aspect with the other group except exposing them to noise, and the noise group animals were subjected to acute noise stress for 4 h only for 1 day (Samson et al., 2006) and their urine samples were collected at the end of stress procedure to analyze the corticosterone level to assess the stress.

Noise stress procedure:

Noise was produced by a loud speaker (15 W), installed at a distance of 30 cm above the cage, and driven by a white noise generator emitting all the frequencies in the range 0–20 kHz. A precision sound level meter was used to set the intensity of sound to 100 dB uniformly in the cage. During the experiment, the noise level peaked at 100 dB immediately after the generator was switched on and continued for 4 hr.



Urine collection procedure:

Urine was collected by placing animals individually in the metabolic cages; all excreta or faeces of rats drop through the bars of the wire mesh floor were filtered and separated so that only the urine could run

through it. So, all urine could be easily collected without contamination with excreta and the sampling was done at 1hr, 4hr and after 24hrs after stress procedure and immediately used for corticosterone measurement.

Assay of corticosterone:

This method was carried out with slight modification from (Singh &Verman 2008) and is based on the oxidation of corticosteroids with ferric iron (III) in acidic medium and subsequent complex with ferrous iron (II) and potassium hexacyanoferrate. 0.5µl of urine was mixed with appropriate volumes of working solutions of corticosterone were transferred into a series of 10 ml volumetric flasks. Sulphuric acid (4N, 2ml) and ferric chloride (0.5%, 2 ml) were added to each followed by potassium hexacyanoferrate (III) solution (0.5%, 0.5 ml). The mixture was heated in a water-bath maintained at 70±2ºC for 30 minutes with occasional shaking and diluted to the 5ml mark with distilled water. The absorbance was measured at 780 nm against the reagent blank.

Eight arm maze:

All behavior parameters were carried after acute stress exposure at time interval of 1 hr, 4hr and after 24hrs along with the controls.

Spatial learning and memory were tested by using a radial eight-arm maze apparatus (Olton et al., 1979). The apparatus, made of gray vinyl chloride plates, had an octagonal central platform, 33.5 cm wide, around which were arranged 60 cm long by 12 cm wide arms. The whole apparatus was elevated 40 cm from the floor in a sound proof chamber. During behavioral training and testing, as food is reward the animals were fasted. Prior to the experiment, a group of animals was trained so that they would become habituated to the apparatus and a piece of cereal was used in the same arms. Initially, animals were allowed to freely explore the maze for 2 consecutive days with all arms baited with cereal. On third day a piece of cereal in four of the eight arms was kept and were trained to locate four food rewards that were always placed in the same set of four arms. The adaptation occurs after a week, each rat was individually housed in a small cage. The adaptation and maze test were performed between 10:00 and 12:00 h. Each individual rat had its own set of four rewarded arms. The room contained several visual reference cues on the wall. For training on the spatial task, only four arms (fixed for that animal) were always baited and food rewards placed at the end of the arms. Each trial began with the placement of the animal on the central platform facing arm number one and ended when the rat had visited the four baited arms or after a period of 10 min.

Y – Maze:

Y-maze was used for the spatial memory as per the reference sited (Conrad et al., 1996). Experiments were performed in a gray polyvinylchloride. The size of each arm was 40 cm long, 15 cm wide, and 35 cm high for rats and 30 cm long, 10 cm wide, and 17 cm high for mice. The floor of the maze was covered with odor-saturated sawdust that was mixed after each trial. The maze was placed in a sound-attenuated room under dim illumination. Numerous visual cues were placed on the walls of the testing room and kept constant during the entire behavioral testing. The test consisted of two trials, separated by a 6-h interval. During the first trial (acquisition phase), one arm of the Y-maze (subsequently called novel arm) was closed with a door, thus allowing the animal to explore only the other two arms, for 5 min for rats and the rats were subjected to noise stress for 4 hr after the interval (retention phase), the animals were allowed to access all the three arms for 5 min. The time spent in each arm of the maze, latency period and triad arm errors were recorded

manually. To avoid manual error and bias a double scoring was done with the help of a fellow researcher. Recognition was assumed to have occurred when the animal spent more time in the novel arm compared with the familiar ones. The total number of arm visits also acts as a measure of general motor activity.

Statistical analysis:

Data are expressed as mean ± standard deviation (SD). All data were analyzed with the SPSS for windows statistical package (version 20.0, SPSS Institute Inc., Cary, North Carolina. Statistical significance between the different groups was determined by independent student T test and the significance level was fixed at $p< 0.05$.

RESULTS

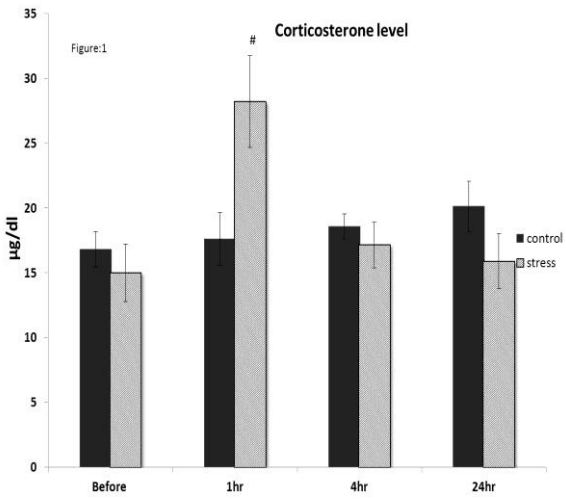
Effect of acute stress on corticosterone levels

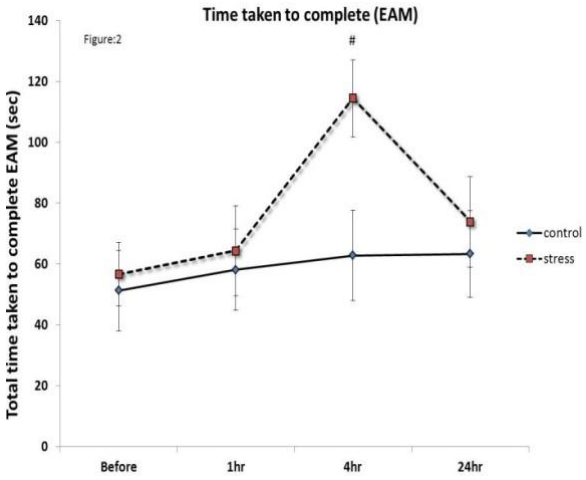
The data are summarized in (Figure: 1) with Mean ±SD, urine corticosterone level was measured in both control and stress group (acute noise stress) after 1 hr, 4 hrs and after 24hrs. The result shows stress exposed rats corticosterone level was significantly increased at 1 hr ($p<0.05$) whereas no significant change in corticosterone level after 4 hrs, and as well as after 24 hrs when compared to control. The data indicated the acclimatization of the body homeostasis after noise stress exposure.

Effect of acute stress on:

Total time taken to complete the eight arm radial maze (EAM):

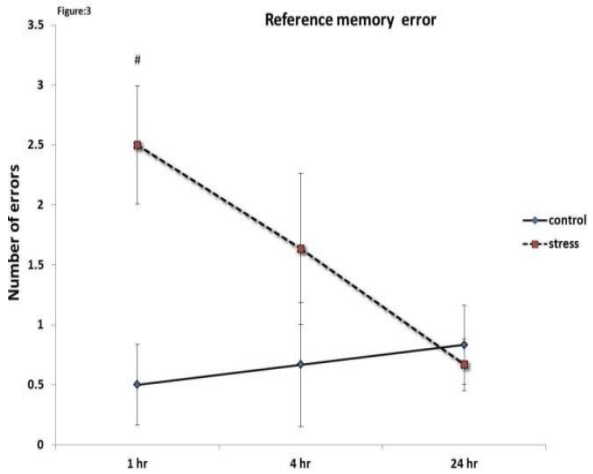
The data are summarized in (Figure: 2) were expressed with Mean ±SD. The time taken by stress exposed animals to complete the eight arm radial maze significantly increased only after 4hrs ($p<0.05$) whereas there was no significant change was observed at 1 hr, and 24hrs, when compared to control.

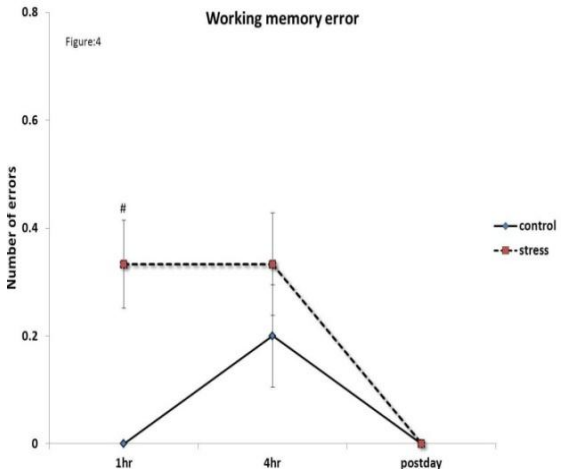




Reference and working memory error:

The data are summarized in (Figure: 3 & 4) with Mean ±SD. Based on Olton's definition (1) Number of reference memory errors, i.e. each entry into a non-baited arm and (2) Number of working memory errors, i.e. re-entries into already visited baited arms were noted down along with the (3) Time taken to visit all the baited arms the scoring was done. Stress exposed animals shows significant increase in both reference and working memory error only at 1hr ($p<0.05$), while in the rest of the durations no significant changes were observed (4hrs and 24hrs) in reference as wells as and working memory error.

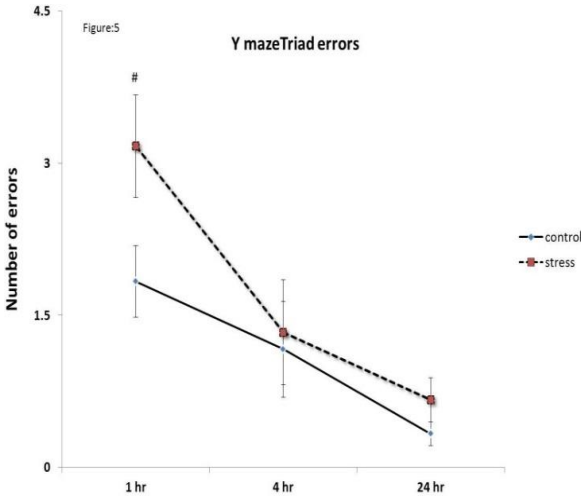




Effect of acute stress on Y maze:

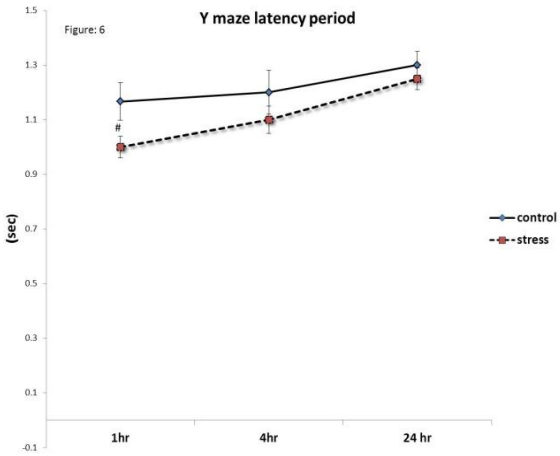
Triad errors:

The data are summarized in (figure: 5) with Mean ±SD. Triad error is given by the wrong sequence and re-entry in the same arm. Stress exposed animals, shows significant change ($p<0.05$) in triad error only at 1 hr, and there was no significant variation was observed at 4hr and after 24 hrs when compare to control.



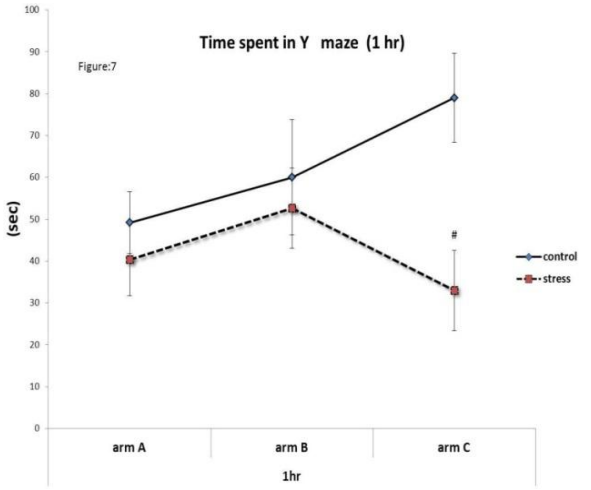
Latency period:

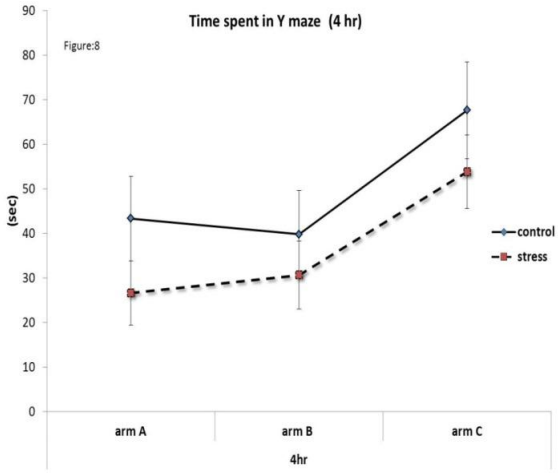
The data are summarized in (Figure: 6) with Mean ±SD. The time taken by the animal to initiate the movement immediately when it placed in the maze. In stress exposed animals, there was significant decrease ($p<0.05$) in the latency period only at 1 hr, and there was no significant latency changes was observed at 4hr and followed even after 24 hrs when compare to control



Time spent in arm A, B and C (Novel arm):

The data are summarized in (figure: 7, 8 & 9) with Mean ±SD. Stress exposed animals, shows a significant decrease at 1 hour ($P<0.05$) in time spent only in arm C (novel arm) and not in arm A and B. However after 4 hour and 24hrs there was no significant variation in time spent in arm A, B and C when compare to control.





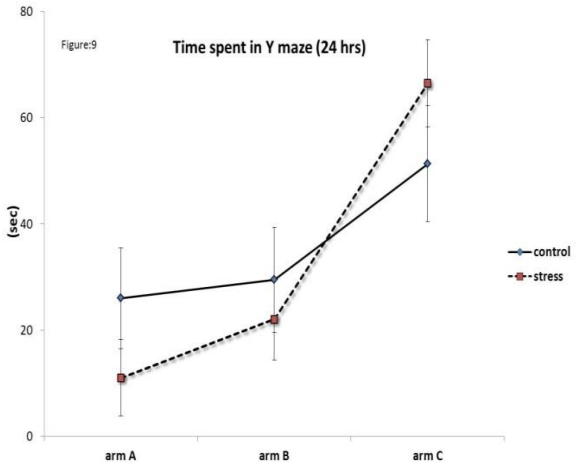


Figure no: 1- 9
Each value represents mean ± SD. Significance at p < 0.05,
 # shows significant change compare with control.
Group -1= control animals, group -2 = acute noise stress exposed animals

DISCUSSION

The analysis of corticosterone metabolites and corticosterone in mice (Spackman & Riley 1978) excreted into urine and feces of mice could offer such a non-invasive technique to assess adrenocortical function. The present study indicates that acute noise memories are affected in the early hours after stress and the effects are normalized by 24 hrs. This was associated with elevated corticosteroid. Y maze normally evaluates hippocampal dependent spatial memory by analyzing exploration of familiar or novel arm location (Gomez and Luine, 2014). In Y maze the triad errors (abnormal arm entry sequence), latency period and time spent in each arm A, B, and C (novel arm) after an acute stressed rats shows significant increase in triad errors and delay in latency period only at 1 hr compare to control and there is no marked changes was observed at 4 hr and on 24 hrs. The stress induced arousal of autonomic sympathetic as well as increased corticosteroid level in the current study may be behind this changes observed and as the level of the stress hormones are normalized then the behavior also become normal. Glucocorticoid administration after memory retrieval impairs later recall (Cai et al., 2006).

In contrast to memory consolidation, memory retrieval seems to be impaired by stress although this could also be interpreted as competitive encoding of new information, related to the stress exposure (Diamond et al., 2007).This acute effect could not be ignored though it is restored to normal level as in normal life human beings are exposed the stress minute to minute. According to Christian Maschke et al., (2000) continuous non-physiological increased level of stress hormones leads to an adverse effect.

Glucocorticoids secreted during stressful episodes can readily cross the blood–brain barrier and act via both mineralocorticoid receptors and glucocorticoid receptors in limbic brain areas (Reul et al., 1985). It is well known that glucocorticoid and catecholamine effects on memory are mediated by the amygdala (McGaugh 2000). Other than its effect on memory, it did not affect the time taken by the stress exposed rats to complete the task in eight arm maze compare to control and this is well supported by (Metz et al., (2005) shows stress and the stress hormone influence motor system function in rats.

Rats with hippocampal lesions are impaired in learning the radial arm (Jarrard, 1993), the T maze (Bannerman et al., 2001). Whishaw, (1998) proposed that the hippocampus is dedicated to monitoring cues generated by self-movement and that it is part of a directional system that provides information to an extrinsic location system. It is a fact that hippocampus provides negative feedback to the HPA axis and has an important role in key aspects of spatial and declarative memory. Corticosterone exerts a concentration-dependent biphasic influence, via selective activation of hippocampal mineralocorticoid and glucocorticoid receptor, on spatial memory (Yau et a., 1995). During stress paradigms in animals, levels of glutamate (Lowry et al., 1993) and serotonin McKittrick and McEwen, 1996) are increased in the hippocampus. Structural changes in the hippocampus associated with chronic stress in animals can be prevented by reducing corticosteroids levels through adrenalectomy. Animal data indicate that corticosteroids are associated with cognitive impairment, cellular changes, and even neuronal death in the hippocampus (Brown et al., 1999).

Memory is not a passive process in which the indiscriminately retain information from the environment. Variables such as context and prior experiences involve in filtering which information has to be retained and the accuracy with which that retention occurs. One important such filter is the emotional state (McEwen and Sapolsky 1995). The amygdala is an essential component of the neural circuitry involved in emotional responses, in general, and in attaching emotional significance to learned stimuli (LeDoux 1993). During the acute noise stress exposure there might be an emotion disturbance also which may interfered with amygdala functions. Anatomically, the amygdala projects to several hippocampal regions (including the CA1 area) (Aggleton 1986) providing various routes by which it may potentially influence hippocampal function. Evidence indicates that the amygdala is critically involved in mediating stress-related effects on behavior and modulating hippocampal function. According to Kim et al., (2001) an intact amygdala is necessary for the expression of the modulatory effects of stress on hippocampal long term potentiation and memory.

Since the corticosteroid elevation is short lived during acute stress the effect on the memory may be reversed in the study. Hence it may be inferred that simultaneous glucocorticoid and noradrenergic activation changes the patterns of brain activity in a way that may contribute to the differential effects of stress on different memory processes (in particular consolidation and retrieval). Normally nature of stress effects on memory is critically timing dependent. As the acute effect the effects are short lived the restoration of memory process could be possible and justify the observation of this study.

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